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Analysis of Family Resemblance. I. Introduction

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In 1969 the staid *Harvard Education Review* published a long report by Arthur Jensen, professor of educational psychology at the University of California, which argued four points: (1) recent attempts to improve the school performance of lower-class American children by compensatory education programs have had little success; (2) their failure is due to genetic differences in cognitive ability between social classes; (3) the observed Negro-white difference in cognitive performance also has a large genetic component; and (4) the last two inferences have implications for educational strategy. Jensen's thesis was violently attacked. Angela Davis called him "that racist professor," and he was subjected to tire slashing and other harassment. Lewontin [1, 2] likened him to Jansen, the seventeenth-century advocate of predestination whose teachings were prohibited by the Inquisition. Lewontin's attack was so vitriolic as to render his attitude toward the Inquisition ambiguous. Our generation has not seen so much intemperance between scientists since Lysenko castigated the bourgeois Mendelists.

Dispassionate criticism of Jensen has concentrated on the first three points: (1) intensive environmental manipulation, as in kibbutz nurseries, may be more effective than the "too little and too late" Project Head Start; (2) the genetic component of social class differences on cognitive tests is not rigorously established; and (3) the genetic evidence is weaker for racial differences in cognition. The first point (which is plausible but far from established) is outside my competence as a geneticist, and in any case has only historical connection with points 2 and 3. Most critics have implicitly accepted Jensen's fourth contention about the implications of the controversy for educational strategy. I have given reasons elsewhere, and will return to them in the Discussion, for denying these implications.

So much for the background of the Jensenist controversy, in which behavioral differences generate more emotion than possible differences in physical or artistic abilities, anthropometrics, or susceptibility to disease, which raise the same methodological problems. It should hardly be necessary to reassert the principle that research which promises to answer interesting or important questions by techniques that pose no hazard to the subjects should be pursued without regard

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for possible abuse of the results by society. Whether current techniques are adequate to determine the genetic component in group differences is a separate question, to which this paper is directed.

FAMILY RESEMBLANCE

Wright [3] found that Burks's data on biological and foster children could not be fitted by path coefficients without assuming a substantially lower heritability of IQ for adults than for children. Because the solution was not unique, it seems desirable to develop an alternative design. Here I shall restrict comparison to the children's generation, although extension to parent-offspring pairs is not difficult. A plausible path-coefficient representation is shown in figure 1. Phenotypes of

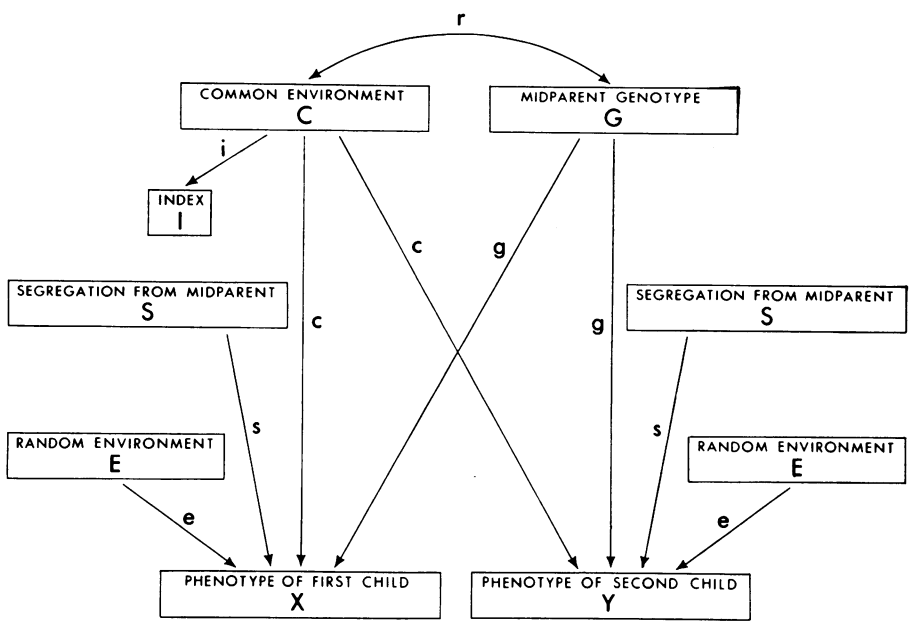

FIG. 1.—A path diagram for sib correlation

two children, X and Y, are resultants of four additive factors: common environment (C), random environment (E), midparent genotype (G), and segregation from the midparent genotype (S). Midparent genotype and common environment are correlated (r), and common environment is imperfectly but linearly measured by an index (I) based on such variables as socioeconomic status, income, parental education, neighborhood, school district, and material and cultural advantages of the home. Construction of such indices will be considered in the next section. The parental genotypes are correlated (m), so the path coefficient from midparent genotype to phenotype of child is $g = h\sqrt{(1 + m)/2}$, where h^2 is the heritability. The path coefficient due to segregation is $s = h\sqrt{(1 - m)/2}$, and $s^2 + g^2 = h^2$.

In the theory of path coefficients, the total variance is constant over all pairs of biological or social relatives with the same path coefficients: $h^2 + c^2 + 2\,grc + e^2 = 1$. This and linearity are critical assumptions for group comparisons.

From figure 1, we have for sibs reared by their own parents

$$\rho = g^2 + c^2 + 2grc. \tag{1}$$

Rao et al. [4] give expectations for other social and biological relationships.

Cattell [5] has elaborated a multiple abstract variance analysis (MAVA). His parameterization includes special environmental components common to identical or fraternal twins and certain covariances between heredity and environment, but neglects interactions and genotypic covariance of mates (m), taking the genetic covariance of half-sibs as half the among-sibship variance (σ_{bh}^2). On his assumptions, $s = g = h/\sqrt{2}$, or as Cattell would write it, $\sigma_{wh} = \sigma_{bh}$.

Jinks and Fulker [6] adapted the biometrical methods of Mather to human behavioral traits. They suggest that "if there is any interaction between genotype and within family environment, then we should find a correlation between the twin sums . . . and the twin differences." Actually, they must mean the absolute value of the twin difference [7]. To have approximate normality, we should take the logarithm of the absolute difference. This test detects only an increase of variance with mean, which can usually be removed by transformation; most interactions and all covariances are missed. Jinks and Fulker provide no specific test for genotype-environment covariance. On the other hand, they give estimates of dominance deviations, which are confounded with environment common to monozygous twins or full sibs but not to parent-offspring pairs or more remote relatives. The notion of dominance deviations for polygenes seems farfetched, since dominance has been shown to decrease with gene effect [8]. Estimation of a variance component due to dominance has not been reliable or useful even in plant genetics, where the method was developed. It is better to treat dominance in terms of major loci, discriminated by complex segregation analysis, with dominance deviations not contributing importantly to resemblance of relatives. Standard errors of variance components are obtained by Jinks and Fulker on the assumption of a normal distribution of variance estimates, which is far from true for moderate sample sizes. The estimation theory should be developed in terms of the z transform of correlation, for which the normality assumption is less restrictive [4]. Clearly, emphasis must be on tests of hypotheses rather than estimation per se.

It might be wondered why I do not estimate the genetic correlation between mates (m) from the phenotypic correlation. The reason is that, as Fisher [9] showed, there are three essentially different structural equations between phenotypic and genetic correlation, depending on whether assortative mating is due to endogamy, direct preference for the trait in question, or preference for a correlated trait. Without capricious assumptions, we cannot directly analyze the phenotypic correlation between mates.

The general rule is that each type of relationship introduces another equation

and another assumption, generally in the direction of overestimating heritability. Until the assumptions are rigorously tested, heritability estimates are unconvincing. While heritability of IQ in man has usually been calculated as greater than .5, by solving equations such as (1), it would not be possible to argue strongly against a smaller value.

As soft as the evidence is within groups, it becomes far worse in group comparisons. Suppose that for two groups A and B we obtain $\rho_A > \rho_B$ for twins or full sibs reared together. This could mean that (1) $h_A^2 > h_B^2$, (2) $c_A^2 > c_B^2$, (3) $m_A > m_B$, (4) $r_A > r_B$, (5) $e_A^2 < e_B^2$, or any combination of these inequalities. Comparison of heritability estimates between groups is ambiguous. Scarr-Salapatek [10], assuming without proof that $r = m = c = 0$, concluded that heritability of IQ decreases with social class in both Negroes and whites. The mean, and possibly the total variance, of IQ also decreases (although the latter is nonsignificant), leading Jensen [11] to suggest a logarithmic transformation, appropriate to multiplicative effects of heredity and environment. Alternatively, if the importance of family environment (c^2) decreases with social class (as might be expected if the relevant lower-class environment is the neighborhood rather than the home), both the estimate of h^2 and the total variance would be diminished. Rao et al. [4] give a more complete discussion of these data.

INDICES

Figure 1 assumes a linear relation of index and family environment. Since the latter cannot be measured directly, formulation of indices is more an art than a science, and any index is a compromise. For example, the U.S. Census places snake charmers in the top occupational category of "professional, technical, and kindred workers" but relegates police officers (along with prostitutes) to the low rank of "service workers." The concept of socioeconomic status (SES), with its medley of income, property, education, and prestige, cannot accurately measure the effect of family environment on behavior or health. As pointed out by Jensen [12] and others, caution must be exercised in applying covariance analysis or stratification to socioeconomic status, because of the possible correlation between SES and genetic factors. On the other hand, substituting an index for environment (i.e., assuming $i = 1$ in fig. 1) will overestimate heritability [3]. Good indices can be constructed by multiple regression of phenotype on family variables.

THE MIXED MODEL

In recent years it has become apparent that segregation analysis should be extended to pedigrees larger than nuclear families and to complex models which include polygenic and oligogenic variability and both quantitative and qualitative information. Charles Smith in Edinburgh, Robert Elston in North Carolina, and our laboratory are working actively on this problem, which promises to throw new light on such diseases as schizophrenia, diabetes, and hyperlipidemia.

The power of genetic studies under this model is improved by replacing qualitative information (like *affected* or *normal*) by a quantitative discriminant of

liability; for example, schizoidia score rather than schizophrenia, and IQ rather than mental retardation. Then recurrence risks can be more reliably predicted with allowance for age, sex, and other covariates.

Among the parameters in this model are polygenic heritability and frequency of major genes [13]. Therefore, segregation analysis within ethnic or social groups is capable of revealing certain differences between groups. The model as currently elaborated neglects environment common to parents and children, so that an estimate of heritability cannot be accepted as purely genetic unless this is supported by path analysis or by tests of consistency among mating types in segregation analysis [4].

The large fraction of mental retardation termed "cultural-familial" is a challenge to epidemiology. It is simplistic to argue either that familial tendency implicates genetic factors or that the higher incidence in lower socioeconomic groups proves environmental determination. A possible approach is to discriminate monofactorial from polyfactorial mental retardation, using only information on probands and their indices, where monofactorial signifies a clear monogenic, chromosomal, or exogenous cause. The first-degree relatives of the monofactorial class should have an elevated frequency of major genes and low heritability as defined, whereas this should be reversed in the pure polyfactorial class, and intermediate in a mixed group. Such studies under incomplete ascertainment should give new insight into the role of major genes in mental defect and their distribution among groups (see [14]) but do not seem capable of determining how much of the remaining "heritability" of cultural-familial mental retardation is due to family environment.

INBREEDING AND OUTCROSSING EFFECTS

Animal and plant experiments show that morbidity increases and size decreases with inbreeding. Studies of inbreeding in man encounter two difficulties: the practical one of separating genetic effects from confounded socioeconomic factors and the theoretical one of explaining the results by major genes or polygenes. Considerable progress has been made in applying covariance analysis and stratification, for example, through sibling controls, although the largest studies on morbidity did not do this [15]. The theoretical problem is more serious. An inbreeding coefficient F does not affect the mean of a trait determined by additive polygenes but increases the variance by a proportion h^2F. On the contrary, rare recessive genes may have large effects on both mean and variance with inbreeding. Polygenes and major genes can in principle be discriminated by complex segregation analysis under the mixed model, whereas correlation and variance component analysis are uninformative.

For deaf mutism, severe mental defect, and limb girdle muscular dystrophy, it has been demonstrated that increased frequency with inbreeding is largely (perhaps entirely) due to rare recessive genes [14]. The evidence for major malformations is suggestive but not overwhelming. Unfortunately, the largest studies of morbidity treated each child as an independent event, without segregation analysis [15].

As an illustration of this point, consider data on 15 children of incestuous mating

who survived early infancy [16]. Of these, two were "severely retarded" and three had IQs less than 70, while the remainder ranged from 85 to 119, with a mean IQ of 105. Clearly, the data are better described in terms of the frequency of mental retardation than by changes in mean and variance. The genetic load agrees with Böök's [17] data on cousin marriages and with the conclusion of Dewey et al. [14] about the role of rare recessive genes in mental retardation, but rarity of sibships of size 2 or more from incestuous matings precludes segregation analysis.

Slatis and Hoene [18] found a significant difference between the distribution of IQ in consanguineous matings and a control. They note, "The two significant differences that have been observed are related to the greater spread of the intelligence scores of the children of consanguineous parents. Two exceptional children were present in this relatively small sample, one with a score of 57 and one with a score of 154. The standard deviation and g_2 for the consanguineous groups would still be higher than for the controls without these two children, but there would no longer be any statistical significance to the differences observed." Polygenes should increase variance but not kurtosis. These observations support the contention that effects of inbreeding on IQ are better described by segregation of rare recessive genes than by polygenes. We cannot extrapolate to effects of maternal inbreeding, which have been little studied.

Outcrossing has an effect which is the converse of inbreeding, a reduction in homozygosity for detrimental recessive genes. Outcrossing may also create novel genotypes, previously unexposed to the sieve of natural selection, which are of reduced fitness. These possibilities were investigated for anthropometrics, mortality, and morbidity among nearly 180,000 births in Hawaii [19]. Heterosis and recombination effects were both nonsignificant. Converted to equivalent inbreeding coefficients by comparison with genetic loads, the random kinship within the same major race (Atlantic or Pacific) was estimated as .0009, and the kinship of members of the same minor race (as Chinese vs. Japanese) was .0005. On the other hand, polymorphic genes gave estimates of .0143 and .1286 by Wahlund's principle. This agrees with other evidence that polymorphisms contribute little to inbred loads [20]. A method to determine kinship from continuous traits has recently been developed [21]. It requires estimation of the genetic covariance matrix within and among traits, and has not yet been applied to racial differences in man.

Such studies could provide predictions of racial differences for behavior. The expected value of the squared difference between two races for a trait with variance σ^2, heritability h^2, and kinship ϕ is [22] $E(D^2) = 4h^2\phi\sigma^2$. For example, if IQ has $h^2 \doteq 1/2$ and $\sigma \doteq 16$, we could expect a racial difference of about $D \doteq \sqrt{E(D^2)} = 16\sqrt{2\phi}$. This is less than one IQ point if kinship is as small as for outcrossing effects in Hawaii but about eight IQ points if kinship is as great as for polymorphisms. It would be interesting to estimate kinship for a wider range of traits in man.

In Hawaii and in other studies [23, 24] there was no evidence of loss of fitness

with recombination. Recently Bresler [25] presented such evidence in a sample of 708 reproductive histories. The data were given as "number of countries of birth" among the great-grandparents of the concepti, with no distinction between F_1 and subsequent mixture (i.e., between heterosis and recombination). Women were asked about their reproductive history and ancestry and that of their husbands. Remembered fetal loss (in any month of gestation) increased markedly with the number of countries represented among the great-grandparents and also with distance between parental birth places.

There are several features of these data which call for comment. Country of birth is a poor guide to ancestry in the United States, since two native-born mates may be more different ethnically than two mates from different countries. No distribution of country of origin is given, but apparently two members of the same ethnic group, one born in the United States and one in Europe, were enumerated as if they were ethnically different. More than one country of birth is recorded for 173 of the fathers but for 254 of the mothers. The difference is highly significant and suggestive of an ascertainment bias. (It will be recalled that women were informants for their husbands.) Bresler did not allow for interfamily variation, nor did he attempt covariance analysis of the important religious and ethnic differences in his material. Early fetal deaths are subject to serious reporting errors. Since the best estimate of the inbred load for all reported fetal deaths is .164 [26], a difference of 1% in fetal loss corresponds to the inbreeding effect expected in first-cousin matings; yet estimates of inbreeding levels in the U.S. are negligible [14]. Since Bresler reported a much larger differential in fetal loss, it cannot plausibly be related to outcrossing. The simplest hypothesis for Bresler's results is that women who underreport their ethnic diversity also underreport their fetal loss. However, this cannot explain the data in his table 5, which indicate that couples born in Rhode Island have much lower rates of reported fetal loss than pairs including at least one member born outside Rhode Island. In view of the small size, puzzling features, and severe methodological problems in this study and its disagreement with larger and better controlled material, its conclusion that "with increased mixture of these gene pools, fetal loss increases proportionately" is unwarranted.

Malécot [22] developed a theory for decline of kinship with geographic distance which allows us to test for inbreeding and outcrossing effects. The theory was extended to metrical traits by Morton [21]. Results for various indicators and predictions from migration and genealogy were consistent in Micronesia [27] and Melanesia [28]. However, discrepancies have been published for Japan and Europe. In the former, contrary to expectation, adult height appears to decrease with distance [29], but this is entirely an artifact of the clinal distribution of stature. In the Swiss canton of Ticino, village exogamy is associated with a 2-cm increase in height [30]. This is remarkable because inbreeding, while retarding rate of growth, has little if any effect on adult height [19, pp. 92–93]. No analysis of covariates which could affect height, such as SES and family size, was attempted. Trevor [31] and Krieger [26] could find no effect of interracial crosses on

metrical traits. Wolanski et al. [32] found no significant association between stature and parental distance in Poland for 16-year-old boys (the sample most comparable to the 18-year-old Tessinois) but an overall tendency for stature, chest circumference, and weight-height index in children to increase with parental distance. They note that "considerable distance, such as between 101–300 and over 300 km, can influence differences in environmental conditions, in the different ecological niches of such distant populations." Careful covariance analysis is necessary to support a genetic interpretation of isolation by distance for traits influenced by the environment.

In many countries there has been a marked increase in body size during the last century. Hultkranz [33] found that the height of Swedish men examined for universal military conscription increased 9 cm in 100 years, with no effect of a severe famine in the 1860s. Height of boys increased 15 cm during a 50-year period, while adult height increased 5 cm [34]. These differences are greater than those between social classes measured at the same time. Dahlberg [35] suggested that increased size is due to isolate breaking, and other investigators have accepted this hypothesis. However, Morton et al. [19] summarized observations which indicate that the increase in size observed during recent generations in Japanese and Western populations is too large and rapid to be attributed to increased heterozygosity through isolate breaking.

HYBRID POPULATIONS

Anthropology pioneered the investigation of hybrid populations. The limited conclusions possible from these studies were reviewed by Morton et al. [19]. The simplest test for a hybridity effect is on total variance, which also depends on the level of environmental stimulation, range of relevant family environment, and gene-environment covariance in the hybrid population. If we neglect these, the variance is expected to decrease in an F_1 hybrid by an amount proportional to $h^2\phi$ (where ϕ is the kinship within parental groups) but to increase in later generations to an extent dependent on diversity of mating types. The possibilities are too complex for a test on total variance to be interpretable.

Various methods to estimate proportions of admixture have been developed [36]. As shown by Balakrishnan [37], data on polymorphisms and metrical traits can be combined by estimating the kinship of a sample of hybrids to each of the parental races. The utility of such estimates in behavior genetics is reduced by any covariance between social rank and visible racial traits, like skin color. Three designs have been suggested to cope with such covariance. In one, the effect of admixture is studied with stratification or covariance adjustment for appearance; this has low power because of the correlation between estimates of admixture from appearance and genetic polymorphisms [36].

Another design looks at racial difference within the same family, using either maternal half-sibs with fathers of different races or adopted children of different race. The hypothesis that parental error, sequential monogamy, or adoption selects differentially within racial groups is difficult to exclude. Apart from this objection,

which applies to all such studies of family resemblance, this design has the advantage of making unnecessary the separation of physical and laboratory evidence on admixture, providing tests are conducted before social rank between foster sibs of different racial appearance is established (presumably at the time of school entry).

The third design uses pairs of sibs from biracial crosses by imposing a variable for midparent race at the top of figure 1, with paths p to common environment and n to race of individual. The latter has path t to an estimate of race and q to phenotype [4]. Then the correlation between racial estimate (M) and environmental index is $r_{MI} = tnpi$, while between racial estimate and phenotype $r_{MY} = t[npc + q]$. On the null hypothesis of no genetic difference between races for the trait in question, $q = 0$. The partial regression of phenotype on true race (N), adjusted for errors of racial estimate and sociological concomitants of race, is $q(\sigma_Y/\sigma_N)$. This is also the estimated genetic difference between the parental races, tested in the environment of the hybrids.

The study of interracial crosses in Hawaii concentrated on tests of outcrossing and recombination effects. Conditions for estimation of parental means from hybrids are more stringent, because relevant environmental differences may persist for generations. However, within some hybrid populations the correlation between ancestry and status is much less than for the parental groups, and in such cases the above analysis should give a more reliable estimate of racial difference than any other method not based on randomization of the environment, which alone would give incontestable evidence.

Jensen [12] recently summarized evidence bearing on Negro-white differences in performance on various tests of cognition and achievement. There is a tendency for the Negro-white differential to be greatest for those tests which are associated with the highest sibling correlation, general intelligence loading in factor analysis, socioeconomic gradient within races, serial correlation within individuals, prestige, financial reward, and (possibly) effect of family environment. While these interesting results do not separate genetic and environmental factors, the more elaborate design discussed above might do so, concentrating on tests which maximally discriminate the groups.

Although the value of genetic studies on racial differences is at best academic, I think a population geneticist has some responsibility to avoid puristic aloofness, lest geneticists conclude that hereditary differences have been proved (or disproved) by psychologists, and psychologists suppose that they have been proved or disproved by geneticists. A minimum standard for the evidence is that it bears the scrutiny of population genetics.

DISCUSSION

In a recent review of human behavioral genetics, I emphasized single gene and chromosomal effects as being most critical for the development of this embryonic science [38]. The brief discussion of group differences concluded that "recent controversy about ethnic differences in behavior is based on two fallacies: first,

that a reliable estimate of heritability can be obtained when the environment is not random; secondly, that heritability is relevant to educational strategy." The present paper does not represent a change in that position. Estimates of h^2, both within and between groups, depend on the credulity of the investigator. The methods discussed here are, I think, better than the classical ones, but they depend on assumptions which have not been, and perhaps cannot be, rigorously tested. More is to be gained from such tests than from estimates of "genetic" parameters. Meanwhile, there is danger that less sensational but more basic research in behavioral genetics may be neglected. One is reminded of Charles Davenport, whose research at the Eugenics Record Office was supported by the Carnegie Institution at a time when they also funded Thomas Hunt Morgan's laboratory. Quite possibly the work in human genetics seemed more socially relevant to the Institution, but at the time *Drosophila* genetics was by any criterion more productive. Behavior genetics does not have at its command methods powerful enough to settle the Jensenist controversy; whether this will prove heuristic or fatal can only be conjectured.

In pursuing this interesting controversy, one would be quite unjustified in claiming that heritability is relevant to educational strategy. The teacher confronted with a neighborhood in which a substantial fraction of the children appear uneducable by either academic or vocational criteria seems to me like a physical therapist treating a case of poliomyelitis: neither need be concerned with the extent to which susceptibility to the observed disorder is genetic. Since the path-coefficient diagram is latticed by a bewildering array of genotype-environment correlations, familial factors are associated with success in school, vocation, and society [39]. However, this correlation is either irrelevant to the success or failure of any novel educational or social policy, or so complexly related that prediction is unreliable. If compensatory education cannot overcome persistent familial differences among schoolchildren, we gain nothing by replacing the word *familial* with *genetic*, *ethnic*, or *cultural*. The argument for multiple streams in education should stand on other grounds than genetics, regardless of how large or small heritability may be shown to be by rigorous methods yet to be developed.

The issue is posed clearly by Scarr-Salapatek [40]. She begins by quoting from me: "Considerable popular interest attaches to such questions as 'is one class or ethnic group innately superior to another on a particular test?' The reasons are entirely emotional, since such a difference, if established, would serve as no better guide to provision of educational or other facilities than an unpretentious assessment of phenotypic differences." She rejoins:

> I disagree. The simple assessment of phenotypic performance does not suggest any particular intervention strategy. Heritability estimates can have merit as indicators of the effects to be expected from various types of intervention programs. If, for example, IQ tests, which predict well to achievements in the larger society, show low heritabilities in a population, then it is probable that simply providing better environments which

> now exist will improve average performance in that population. If h^2 is high but environments sampled in that population are largely unfavorable, then (again) simple environmental improvement will probably change the mean phenotypic level. If h^2 is high and the environments sampled are largely favorable, then novel environmental manipulations are probably required to change phenotypes, and eugenic programs may be advocated.

Her point of view seems unrealistic in two respects. First, it assumes that heritability estimates derived without randomization of the environment are reliable. Second, it neglects the enormous expenditure devoted to social and educational experiments. There is no need to predict, from doubtful estimates and simplifying assumptions, what the effect might be of an experiment that is going to be tried anyhow. The more conspicuous abscesses in American society involve populations in unfavorable environments. Scarr-Salapatek's syllogism states that environmental manipulation is worth trying in such a situation, regardless of h^2. How can she then argue that estimation of h^2 is useful as a guide to "intervention strategy"?

I think that all such arguments are based on subjective reality. One of the deepest, and therefore most irrational, tendencies in American society is to base the claim for social justice on biological equality. If this were questioned, as meritocracy has been questioned, might the social consequences be unfavorable? The nearest approach to an answer may be Herbert Spencer's remark quoted by Jensen: ". . . the ultimate infidelity is the fear that the truth will be bad." If that be so, we may proceed dispassionately with the rationale of Otto Klineberg: "There is no scientifically acceptable evidence that ethnic groups differ in innate abilities. This is not the same as saying that there are no ethnic differences in such abilities." To this I add the conviction that when such differences are incontrovertibly established, perhaps by neglecting more basic questions, it will be an academic triumph for behavior genetics, with no relevance to rational social policy. By rational I mean a policy that attempts to maximize the value of an individual, choosing methods according to their phenotypic effects without regard to heritability of individual or group differences.

SUMMARY

Designs to study family resemblance, construct environmental indices, separate polygenes and major loci, and analyze inbreeding and outcrossing effects and hybrid populations are discussed. Recent evidence and an assessment of the implications of such studies for the determination of the heritability of human traits such as IQ are reviewed.

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